

IN THE UNITED STATES PATENT AND TRADEMARK OFFICE

Applicant	:	Weinberger et al.
Appl. No.	:	10/789,169
Filed	:	February 27, 2004
For	:	EFFECT OF BDNF GENOTYPE ON HIPPOCAMPAL FUNCTION AND VERBAL MEMORY AND RISK FOR SCHIZOPHRENIA
Examiner	:	Sitton, Jehanne Souaya
Group Art Unit	:	1634

DECLARATION UNDER 37 CFR 1.132 OF DANIEL R. WEINBERGER, M.D.

I, Daniel R. Weinberger, M.D., do hereby declare:

1. Introduction

I am a named inventor of the above-identified application. A true and correct copy of my Curriculum Vitae is attached as Exhibit 1.

2. Compliance with 35 USC 112/1 enablement

I understand that the United States Patent and Trademark Office (USPTO) rejected the claims under 35 USC 112/1 as failing to meet the enablement requirement. I understand that the test for enablement is whether one skilled in the art could make or use the subject matter defined by the claims without undue experimentation. I understand that the “Wands” factors (the nature of the invention and breadth of the claims, the amount of direction/guidance and presence/absence of working examples, the state of the prior art and the predictability or unpredictability of the art, the level of skill in the art, and the quantity of experimentation needed to make or use the invention) are to be considered in determining whether any necessary experimentation is undue. Here, the specification is enabling with respect to the claimed subject matter. Thus, here, considering all the factors related to the enablement issue, it must be concluded that the specification is enabling with respect to the claims at issue.

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3. Nature of the invention and breadth of the claims:

a. Claims 1 and 2,

and claims dependent thereon, are related to methods for predicting the likelihood that a human will have impaired or enhanced hippocampal function, or hippocampal dependent verbal memory, comprising the steps of obtaining a DNA sample from a human to be assessed and determining the presence or absence of a single nucleotide polymorphism from G to A resulting in the substitution of a methionine residue for a valine residue at amino acid position 66, relative to the start of the precursor protein sequence for brain-derived neurotrophic factor (BDNF), where a single nucleotide polymorphism from G to A resulting in the substitution of a methionine residue for a valine residue at amino acid position 66 (relative to the start of the precursor protein sequence) is indicative of the likelihood that a human will have impaired hippocampal function, or hippocampal dependent verbal memory, relative to valine, and a single nucleotide polymorphism from A to G resulting in the substitution of a valine residue for a methionine residue at amino acid position 66 (relative to the start of the precursor protein sequence) is indicative of the likelihood that a human will have enhanced hippocampal function, or hippocampal dependent verbal memory, relative to methionine.

b. Claims 13 and 14,

and claims dependent thereon, are related to methods for predicting the likelihood than a human will have impaired or enhanced hippocampal function, or hippocampal dependent verbal memory, comprising the steps of obtaining a biological sample from a human to be assessed containing the precursor BDNF protein or relevant portion thereof and determining the amino acid present at amino acid position +66 relative to the first amino acid of the precursor protein, wherein the presence of methionine at this position is indicative of the likelihood that a human will have impaired hippocampal function, or hippocampal dependent verbal memory, relative to valine, and the presence of valine at this position is indicative of the likelihood that a human will have enhanced hippocampal function, or hippocampal dependent verbal memory, relative to methionine.

4. **The amount of direction/guidance & presence/absence of working examples:**

a. **Verbal Memory**

i. **Specification at paragraph [0008]**

The specification teaches that BDNF is a neurotrophin and that the BDNF gene contains at least one known nonconservative SNP producing a met66val substitution. The specification teaches that schizophrenia appears to involve hippocampal abnormalities, including deficits in verbal memory. The specification teaches that verbal memory deficits are also found in unaffected sibs of patients, suggesting a genetic trait related to susceptibility. The specification teaches that we hypothesized that the met66val polymorphism would affect verbal memory. The specification teaches that verbal memory was assessed in 184 patients with schizophrenia, 283 siblings, and 101 controls. The specification teaches that the effect of genotype was significant across all groups for memory scores ($p < 0.008$) and that the rarer met allele was associated with poorer performance. The specification teaches that BDNF genotype had no effect on IQ or prefrontal cognitive measures.

ii. **Egan et al., Cell 112:257 (Jan 2003)**

I understand that the rule according to MPEP 2164.05 is that an applicant may provide a declaration after the filing date that demonstrates that the claimed invention works as claimed. The post-filing date art Egan et al., Cell 112:257 (Jan 2003), of record, is such a demonstration. Egan et al. 2003 provides that, despite substantial progress in animal studies, BDNF's relevance in human memory and hippocampal function has not been examined directly. In Egan et al. 2003, we examined the effects of a valine (val) to methionine (met) substitution in the 5' pro-region of the human BDNF protein. While we found no relationship between BDNF genotype and schizophrenia, our results demonstrate that BDNF plays a role in hippocampal function and verbal memory in humans.

In a first experiment, we examined the effects of BDNF genotype on measures of verbal memory in a cohort of 641 subjects, including normal controls, patients with schizophrenia, and their unaffected siblings, using measures from the Wechsler Memory Scale, revised version (WMS-R), a test of verbal memory. Delayed recall scores from the WMS-R reflect the amount

of information from two stories with 50 total elements that subjects are able to recall following a 0.5 hr delay. Patients with schizophrenia had substantially lower scores compared to controls, while siblings were intermediate between these groups, consistent with earlier reports (Egan et al., *Proc. Natl. Acad. Sci. USA* **98**: 6917, 2001, of record; Figure 1A). In the entire sample, BDNF genotype had a significant effect on these memory scores ($F = 3.89$, $df = 2$, 591, $p = .02$). In the group of 133 controls alone, BDNF genotype also had a significant effect on memory scores ($F = 5.04$, $df = 2$, 130, $p = .008$). While including patient and sibling groups did not substantially add to the results, both groups showed the same effect seen in controls with met/met subjects tending to score lower than other genotype groups (Figure 1A). Post hoc comparisons in the normal subjects alone showed that met/met homozygotes had lower scores compared to val/val ($p < .005$) and val/met ($p = .052$), while in the entire cohort, met/met homozygotes had lower scores compared to the other two genotype groups ($p = .007$). Within each group (controls, siblings, patients), each genotype group was well matched on a variety of demographic parameters (see Figure 1B), suggesting they did not account for the effect of BDNF genotype. Similar effects of BDNF genotype were seen with immediate recall scores (of the stories described above) from the WMS-R (e.g., normal group, $F = 3.65$, $df = 2$, 130, $p < .03$). Post hoc comparisons again showed that met/met homozygotes had lower scores compared to val/val ($p < .01$) and val/met ($p = .07$) groups.

BDNF genotype had no significant effect in either the normal subjects or the entire cohort on a second memory test which required recall of word lists, the California Verbal Learning Test (CVLT), immediately after hearing the list (1–5 summary scores, $F = 0.79$, $df = 2$, 560; $p = .45$; post hoc comparisons $p > .14$) and again 20 min later (long delay free recall scores, $F = 0.23$, $df = 2$, 560; $p = .79$; post hoc comparisons $p > .37$). The CVLT is considered to have a larger “prefrontal component” (Kopelman et al., *Brain* **121**: 875, 1998, of record) compared to the WMS-R, thus excluding it as a test of verbal memory that is hippocampal dependent, and leaving the WMS-R as a valid test of hippocampal dependent verbal memory. No effect of BDNF genotype was seen on other types of memory, such as semantic memory ($F = 0.02$, $df = 2$, 591; $p = .98$) or working memory/executive function (WCST, $F = 0.33$, $df = 2$, 590; $p = .71$) (Figure 1B).

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These results indicate that the val66met polymorphism exerts its most robust effects on hippocampal dependent verbal memory.

b. IQ and Prefrontal Cognitive Measures

Our results also speak to a current debate about the genetics of human intelligence. Specific cognitive abilities are partially correlated, and the extent of this correlation is referred to as “g”, synonymous with IQ. Much genetic epidemiological data suggest that the genetic determinants of different cognitive abilities largely overlap with each other and with IQ. In other words, genes influence specific cognitive traits by virtue of their “top down” effects on g. The alternative “bottom up” view that genes contribute to IQ primarily through their unique and more direct effects on specific cognitive modules has found little support. The present data are consistent with the latter formulation. We did not detect an effect of BDNF on IQ or any other cognitive ability. A recent study of the role of the COMT gene on working memory and prefrontal physiology, but not IQ, provides further evidence for this view (Egan et al. 2001, above). Our current data suggest that the effects of BDNF and COMT are somewhat specific and mediated through “modular” cognitive elements.

c. Hippocampal Function

i. Specification at paragraph [0008]

The specification teaches that schizophrenia also appears to involve abnormal patterns of hippocampal activation during memory tasks assessed with functional magnetic resonance imaging (fMRI). The specification teaches that abnormal hippocampal activation is also found in unaffected sibs of patients. The specification teaches that, in two separate cohorts studied with fMRI, subjects with a met allele had abnormal patterns of hippocampal activation while performing memory tasks, compared to val/val homozygote subjects.

ii. Egan et al., Cell 112:257 (Jan 2003)

The post-filing date art Egan et al., Cell 112:257 (Jan 2003), of record, demonstrates that, with regard to this additional aspect of the invention, the claimed invention works as claimed. To further characterize the effects of BDNF genotype, we performed an in vivo assay of hippocampal physiology using the blood oxygenation level dependent (BOLD) fMRI technique

in subjects performing the N-back working memory task. The brain network subserving the N-back task (which is not a test of verbal memory per se) is thought to involve primarily neocortical regions, particularly dorsolateral prefrontal cortex. However, these tasks also produce a robust and reliable deactivation or disengagement of the hippocampus. Hippocampal deactivation has been reported to be disrupted in clinical pathological states. We hypothesized, therefore, that the BDNF met allele would disrupt the normal hippocampal fMRI disengagement pattern during performance of the N-back working memory task.

We studied two independent cohorts of healthy subjects (patients were not included), each comprised of val/met and val/val genotype subgroups; no met/met subjects were included due to their low population frequency (< 5%). The members of the cohorts were selected so that N-back performance, age, and gender did not differ between the genotype subgroups. In the first cohort, val/met individuals had an abnormal pattern of increased activation of bilateral caudal hippocampus. In contrast, val/val subjects showed the characteristic hippocampal deactivation pattern. Directly comparing val/val and val/met subjects showed this difference to be statistically significant (see Figure 2). More specifically, restricting the analysis to the medial temporal lobes revealed two clusters of activation showing the inappropriate overactivation of val/met subjects in bilateral hippocampus that were significant at the set level ($p < 0.05$, $c = 2$) (Figure 2A). To replicate this unusual finding, we examined a second cohort of healthy subjects (patients not included). Val/met subjects again showed an abnormal pattern of increased bilateral hippocampal activation compared to baseline while val/val subjects again showed deactivation. Direct comparison of the two genotype groups again showed significant differences in hippocampal locales (Figure 2B).

Because these healthy subjects were matched prior to the experiment on task performance to remove this as a potential confounder, it cannot account for these findings (Figure 2C). Within each cohort, genotype groups were also matched on demographic variables, suggesting they did not account for the BDNF effects. One exception was a slight difference in full scale IQ and WMS-R scores in the second cohort (Figure 2C). However, adding IQ as a covariate again revealed significant bilateral clusters of differential caudal hippocampal activation. To ensure the differences between groups were not due to uneven sample sizes, all analyses were also

performed using subsets with the same number of subjects in each genotype group. The results were essentially identical. Scatter plots did not reveal outliers. In summary, in two cohorts we found abnormal activation of hippocampus in val/met subjects compared to the normal pattern of deactivation in val/val subjects. Although this was seen in the context of a working memory task, which is typically more dependent on prefrontal function, and although the mechanism by which val66met might produce this effect is uncertain,¹ the data indicate that the BDNF met allele relative to val is associated with a qualitatively altered hippocampal response.

d. Extension to Different Ethnicities

Although I understand that the USPTO takes the position that in previous studies investigating the association between BDNF genotype and neuropsychiatric disorders, northern Europeans appear to be much more affected than Asian populations, suggesting that some ethnicities may compensate for the variation in the BDNF gene, because we see identical effects in one ethnic group alone (European Americans) and because similar effects are seen in both the normal controls and our schizophrenia families, said ethnicities should be equally affected by the presence of this polymorphism.

5. The state of the prior art and the predictability or unpredictability of the art:

Although I understand that the PTO takes the position that the BDNF polymorphism has been linked to altered susceptibility to various neuropsychiatric disorders, such as Alzheimer's disease, Parkinson's disease, depression, eating disorders, and bipolar disorder, in conclusion, we did not find evidence that BDNF was associated with increased risk for schizophrenia, nevertheless, because this polymorphism has significant effects on verbal memory and hippocampal activation, the met allele may affect other human illnesses, including Alzheimer's disease, head trauma, various psychiatric conditions, and even normal aging.

¹ I understand that the rule according to MPEP 2164 is that it is not a requirement of patentability that an inventor understand how or why the invention works.

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6. The level of skill in the art:

I understand that according to *Amgen Inc. v. Hoechst Marion Roussel Inc.*, 57 USPQ2d 1449, 1518 (D. Mass. 2001), the level of skill in the molecular biology art was considered that of a postdoctoral fellow working in the laboratory. Thus, the level of skill in the art was high.

7. The quantity of experimentation needed to make or use the invention:

At the time of the 31 August 2001 priority date, for guidance regarding such conditions, refer to, for example, Sambrook et al., 1989, *Molecular Cloning, A Laboratory Manual*, Cold Spring Harbor Laboratory Press, New York; and Ausubel et al., 1989, *Current Protocols in Molecular Biology*, Green Publishing Associates, Inc., and Wiley & Sons, Inc., New York.

Verbal Memory: Neuropsychological tests were known in the art, e.g., Egan et al., *Biol. Psychiatry* 50: 98 (Nov 2001), of record, and Weickert et al., *Arch. Gen. Psychiatry* 57: 907 (2000), of record.

Hippocampal Function: Functional magnetic resonance imaging was known in the art, e.g., Callicott et al., *Cereb. Cortex* 10: 1078 (Nov 2000), of record, and Ogawa et al., *Proc. Natl. Acad. Sci. USA* 89: 5951 (1992), of record.

Antibodies that specifically recognize the BDNF valine66 but not the BDNF methionine66 variant: Refer to, for example, Harlow and Lane, 1988, *Antibodies, A Laboratory Manual*, Cold Spring Harbor Laboratory Press, New York, describing how an antibody may be raised against a unique peptide sequence by synthesizing the peptide, conjugating it for immunization, immunizing rabbits, purifying the antibodies against the immunizing peptide, and examining the specificity of antibodies by western blot, all as shown by Zhou et al., *J. Neurochem.* 91: 704 (2004), of record, in which an antibody is produced that specifically recognizes the precursor BDNF, but not mature BDNF, by western blot. Similarly, one possessing the ordinary level of skill in the pertinent art at the time the invention was made could produce an antibody that specifically recognizes the amino acid present at amino acid position +66 relative to the first amino acid of the precursor BDNF protein for determining the presence of methionine or valine at this position.

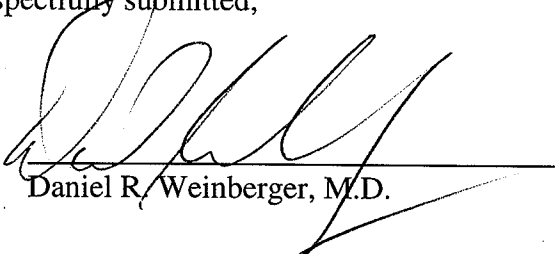
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I declare that all statements made herein of my own knowledge are true and that all statements made on information and belief are believed to be true; and further that these statements were made with the knowledge that willful, false statements and the like so made are punishable by fine or imprisonment, or both, under Section 1001 of Title 18 of the United States Code and that such willful, false statements may jeopardize the validity of the application or patent issuing therefrom.

Respectfully submitted,

Dated: 6/14/07

By:


Daniel R. Weinberger, M.D.

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EXHIBIT 1

Updated 12/8/06

CURRICULUM VITAE

Daniel R. Weinberger, M.D.

Director, Genes, Cognition and Psychosis Program, DIRP, NIMH
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Bethesda, MD 20892
weinberd@mail.nih.gov



Education:

1969-B.A. The Johns Hopkins University
1973-M.D. The University of Pennsylvania

Professional Positions:

- 2003- Director: Genes, Cognition and Psychosis Program, Intramural Research Program, National Institute of Mental Health (NIMH), National Institutes of Health (NIH), Bethesda, MD 20892

- 1998- Chief, Clinical Brain Disorders Branch, Intramural Research Program, NIMH, NIH Bethesda, MD 20892

- 1986-1998 Chief, Clinical Brain Disorders Branch, Intramural Research Program, National Institute of Mental Health (NIMH), Neuroscience Center at Saint Elizabeths, Washington, D.C. 20032

- 1983-1988 Director, Behavioral Neurology Service, Saint Elizabeths Hospital, Washington, D.C.

- 1983-1986 Director of Movement Disorder, Dementia Clinic, Experimental Therapeutics Branch NINDS, NIH, Bethesda, Maryland.

- 1983-1986 Chief, Section on Clinical Neuropsychiatry and Neurobehavior, Neuropsychiatry Branch, Intramural Research Program, National Institute of Mental Health (NIMH), Saint Elizabeths Hospital, Washington, D.C.

- 1981-1982 Head, Clinical Neuropsychiatry and Neurobehavior Unit, Adult Psychiatry Branch, Intramural Research Program, NIMH, Saint Elizabeths Hospital.

- 1977-1981 Staff Psychiatrist, National Institute of Mental Health.

1977-1978 Research Ward Director, Adult Psychiatry Branch, Intramural Research Program, National Institute of Mental Health.

Professional Training:

1980-1983 Resident in Neurology, George Washington University Medical Center, Washington, D.C.
1976-1977 Chief Resident, Massachusetts Mental Health Center, Boston, Massachusetts.
1975-1976 Associate in Medicine (Psychiatry Division), Peter Bent Brigham Hospital, Boston, Massachusetts.
1974-1976 Resident in Psychiatry, Massachusetts Mental Health Center, Boston, Massachusetts.
1973-1974 Intern (R-1, Medicine), UCLA L.A. County-Harbor General Hospital, Torrance, California.

Academic Appointments:

1995 Clinical Professor of Psychiatry and Neurology, George Washington University, School of Medicine, Washington, D.C.
1993-1995 Clinical Professor of Psychiatry, Ohio State University School of Medicine, Columbus, Ohio.
1984-1994 Associate Clinical Professor of Neurology and Psychiatry, George Washington University School of Medicine, Washington, D.C.
1982-1984 Associate Clinical Professor of Psychiatry, George Washington University School of Medicine, Washington, D.C.
1978-1981 Assistant Clinical Professor of Psychiatry, George Washington University.
1974-1977 Clinical Fellow in Psychiatry, Harvard Medical School.
1973-1974 Graduate Fellow in Medicine, UCLA School of Medicine.

Additional Professional Activities:

1999-2002 Founder, Director, Consultant – Biognosis, US, Inc. – A translational genomics biotechnology company
1995-1999 Founder, Director, Contemporary Biobehavior Research, LLC. - A clinical trials SMO. Sold 3/99 to Comprehensive NeuroScience, Inc.
1978- General Psychiatric and Neurologic Practice (Part-time), 5415 Connecticut Avenue, Washington, D.C.
1979-1990 Examiner, American Board of Psychiatry and Neurology.
1974-1977 Emergency Room Physician, Cardinal Cushing General Hospital, Brockton, Massachusetts.
1974-1976 General Medical Practice (Part-time), Bridgewater Medical Center, East Bridgewater, Massachusetts.

Medical Specialty Board Certification:

1978-American Board of Psychiatry and Neurology (Psychiatry)
1984-American Board of Psychiatry and Neurology (Neurology)

Journal Editorial Boards:

1986-2006 Biological Psychiatry (Associate Editor, 1997)
1987- International Journal of Schizophrenia Research
1987- Journal of Neuropsychiatry and Clinical Neuroscience
1987- 2004 Psychiatry
1989-1996 Progress in Neuropsychiatry and Psychopharmacology
1990- Psychiatry Research: Neuroimaging
1990- Journal of Psychiatry and Neuroscience
1991-2006 Neuropsychopharmacology

1991-	<u>Development and Psychopathology</u>
1992-2001	<u>Harvard Review of Psychiatry</u>
1994-	<u>Cognitive Neuropsychiatry</u>
1994-2002	<u>Synapse</u>
1995-2000	<u>Frontiers in Bioscience</u>
1996-1999	<u>Journal of Neural Transmission</u> (Section Editor)
1996-2001	<u>Neuroscience.Net</u>
1996-2002	<u>Lancet Neurology Network</u> (Associate Editor)
1997-2002	<u>Annual Review of Medicine</u>
1999-	<u>Current Psychiatry Reports</u>
2000-	<u>Psychopharmacology Bulletin</u>
2000-	<u>Journal of Clinical Psychopharmacology</u>
2001-	<u>NeuroImage</u>
2002-	<u>NeuroRx</u>
2003-	<u>Neuroscience Letters</u> (Associate Editor)

Honors and Awards

Phi Beta Kappa
 Alpha Omega Alpha
 A.E. Benett Foundation Award for Clinical Science (1981), Society of Biological Psychiatry
 Morton Prince Award (1984), American Psychopathological Association
 Judith B. Silver Award (1985), National Alliance for the Mentally Ill
 Arthur S. Flemming Award (1986), Washington Jaycees, for outstanding government service
 Meritorious Service Medal (1987), United States Public Health Service
 Joel Elkes International Award (1989), American College of Neuropsychopharmacology
 Foundations Fund Prize for Research, American Psychiatric Association (1991)
 Distinguished Service Medal, United States Public Health Service (1992)
 Lieber Award, NARSAD, (1993)
 Dean Award, American College of Psychiatrists (1994)
 Gold Medal Award, Society of Biological Psychiatry (1994)
 Nathaniel Winkelman Award (1994), Einstein Medical Center, Philadelphia
 Kempf Fund Award (1995) American Psychiatric Association
 British Medical Association Book of the Year Award (1996)
 World Federation of Societies of Biological Psychiatry Research Prize (1997)
 Elected into the Institute of Medicine, National Academy of Sciences (1999)
 Adolf Meyer Award, American Psychiatric Association (2000)
 Robert Sommer Medal, Justus Liebig University School of Medicine, Germany (2002)
 Warren Foundation Award (2003) International Society of Schizophrenia Research
 Russel Bullyea Award (2005), National Alliance for the Mentally Ill (inaugural recipient)
 G. Burroughs Mider Award (2005), NIH Director
 Roche-Nature Medicine Award for Translational Neuroscience (2006) (inaugural recipient)

Selected Honorific Lectures

1986	Marshall University School of Medicine Distinguished Lecturer
1987	University of Oklahoma School of Medicine AOA Graduation Address
1988	Roerig Visiting Professor – University of Washington, Seattle
1990	The Public Lecturer - Society for Neuroscience, 20th Annual Meeting, St. Louis
1990	Roerig Visiting Professor - University of New Mexico
1991	Roerig Visiting Professor – University of Michigan
1992	Neal Mysell Lecture - Harvard Medical School
1995	First Memorial James Cleghorn Lecture - McMaster University School of Medicine

- 1999 Pfizer Visiting Professor – Baylor University
- 1999 Harold Cooper Lectureship – University of Texas, Houston
- 2000 Allene Rubin Memorial Lecture – Johns Hopkins University School of Medicine
- 2001 The Field Lecture – University of Minnesota School of Medicine
- 2002 Thomas L. O'Donohue Memorial Lecture in Neuropharmacology – Howard University College of Medicine
- 2003 Abraham Ribicoff Lecture – Yale University School of Medicine
- 2003 Sir Thomas Finch Memorial Lecture and Visiting Professor, University of Sheffield College of Medicine, England
- 2003 Paul Jansen Lecture, Institute of Psychiatry, London
- 2003 Thomas Smith Lecture, National Institute of Psychobiology, Hebrew University, Jerusalem
- 2005 The G. Burroughs Mider Lecture – NIH
- 2007 Williams B. Abrams Lecture – FDA

Institution and Industry Advisory Boards

- 1990 Alzheimer Disease Foundation
- 1993 Adams Super-Center for Brain Studies, Tel Aviv University
- 1993 Sandoz Advisory Board (Clozapine)
- 1995 Jansen Advisory Board (Risperidone)
- 1995 Abbott Advisory Board (Sertindole)
- 1996 Zeneca Advisory Board (Seroquel)
- 1996 Pharmacia and Upjohn CNS Advisory Board
- 1996 Knoll Pharmaceuticals CNS Advisory Board
- 1997 NARSAD Council
- 1997 Eli Lilly Neuroscience Advisory Board
- 1998 Karolinska Institute CNS Advisory Board
- 1999 Lieber Center, Columbia University
- 2000 Astra-Zeneca CNS advisory board
- 2001 Domenici Institute for Mental Illness/Neuroscience Discovery (MIND) Scientific Advisory Board
- 2002 NAMI Scientific Council
- 2004 Ohio State Program in Pharmacogenetics

Organizations (offices held):

- American Medical Association
- American Psychiatric Association
- Washington Psychiatric Society
- American Association for the Advancement of Sciences
- Society of Biological Psychiatry - (President 1998-99)
- American Academy of Neurology
- Behavioral Neurology Society
- Washington Neurology Society
- American College of Neuropsychopharmacology (fellow 1991)(President 2005)
- Society for Neuroscience
- American Neuropsychiatric Association
- American Neurological Association
- American College of Psychiatrists (Fellow 2001)

Selected Committees

- 1999-2002 Chairman, Scientific Committee, World Federation of Societies of Biological Psychiatry
- 1999-2001 Chairman, NIH 3T Users Committee

1998-2001 NIH Central Tenure Committee
 1998-1999 NIH Clinical Research Revitalization Committee
 1995-1998 Elected to Executive Council, American College of Neuropsychopharmacology
 1995-1998 Elected to Executive Council, Winter Conference on Brain Research
 1994-1997 Scientists Promotion Review Committee, NIMH
 1993-1995 Scientific Program Committee, American Academy of Neurology
 1993-2000 Steering Committee, NIH In Vivo NMR Center
 1985-1988 Elected to Council of the Assembly of Scientists-NIMH/NINDS

NIH Extramural Grant Mentorship/Consulting

Numerous RO-1 and K awards
 Yale NIMH Clinical Research Center
 University of Pittsburgh NIMH Neuroscience Center
 University of North Carolina NIMH Clinical Research Center
 Mt. Sinai MIRECC
 Baylor University MIRECC

Military Service:

1977Lt. Cdr. (0-4) USPHS
 1982Cdr. (0-5) USPHS
 1986Cpt. (0-6) USPHS (exceptional capability promotion)

Patents

- 1) Neonatal Ventral Hippocampal Excitotoxic Lesion as a Rodent Model of Schizophrenia for Testing and Developing Antipsychotic Treatments (domestic and international patents (U.S. patent No. 07/967,367)
- 2) A Simple and Rapid Method to Prepare Optically Pure 3-Quinuclidinyl-4-[123I]iodobenzilate Isomers from Stannylate QNB Precursor as a SPECT Imaging Agent for Muscarinic Acetylcholine Receptors (U.S. patent No. 08/229,837)

Non-Federal Grant Support

NARSAD Young Investigator Awards:

- 1989-90 - Neuroanatomical and neuropathological analysis of mesencephalic dopamine systems in schizophrenics - Hyde TM
- 1989-91 - The neurobiology of cognitive impairment in schizophrenia - Gold JM
- 1990-92 - The role of the medial prefrontal cortex in the action of atypical antipsychotic drugs - Jaskiw GE
- 1994-96 - Genetic determinants of nigrostriatolimbic dopamine system responsiveness to pharmacological and stressful stimuli in rats with neonatal hippocampal damage - Lipska BK
- 1997-99 - Cortical glutamate neurons in schizophrenia and affective disorders – Bachus S
- 1999-01 - Non-water suppressed proton magnetic resonance spectroscopy in schizophrenia – Bertolino A
- 1999-01 - Novel intermediate phenotypes in schizophrenia: Functional MRI and the hippocampus – Callicott JH

- 1990 - NARSAD Established Investigator Award - "Development for Clinical Studies of Novel Neurochemical Imaging Agents"
- 1991 - Tourette's Foundation Grant - "Tourette's Syndrome in Monozygotic Twins"
- 1994 - CRADA - Neuromedica - "Therapeutic and Diagnostic Research Using a Novel Carrier System..."
- 1995 - Stanley Foundation Grant - "3-D fMRI Mapping of Working Memory in Patients with Schizophrenia and their Siblings: A Genetic Linkage Study"
- 1997 - CRADA - Cortex Pharmaceuticals - "Clinical Investigations of Novel Glutamate Potentiating Drugs"
- 2000 - NARSAD Distinguished Investigator Award - "Biological Traits and the Genetics of Schizophrenia"
- 2000 - CRADA - NPS Allelix - "Effects of Inhibitors of Glycine Transporters on Behavioral Deficits in the Animal Model of Schizophrenia"
- 2001 - CRADA - Genset, S.A.. - "Brain Expression Studies of a Novel Gene Related to Psychosis"
- 2002 - CRADA - Yamanouchi Pharmaceutical Co. Ltd. - "Determination of Expression of Novel G-protein Coupled Receptor Family SREB in Normal and Diseased Human Brain"

PUBLICATIONS IN PEER-REVIEWED SCIENTIFIC JOURNALS

1. Cherubin CE, Kane S, **Weinberger DR**, Wolfe E and McGinn T: Persistence of Transaminase abnormalities in former narcotic addicts. Ann Int Med 76:385-389, 1972.
2. **Weinberger DR** and Kelly MJ: Catatonia and malignant syndrome: A possible complication of neuroleptic administration. J Nerv Ment Dis 165:263-268, 1977.
3. Greenblatt DJ, Shader RI, **Weinberger DR**, Allen MD and MacLaughlin OS: Effect of a cocktail on diazepam absorption. Psychopharmacology 57:199-203, 1978.
4. Carman JS, Gillin JC, Murphy DL, **Weinberger DR**, Kleinman JE, Bigelow LB and Wyatt RJ: Effects of a selective inhibitor of type A monoamine oxidase--Lilly 51641--on behavior, sleep and circadian rhythms in depressed and schizophrenic patients. Commun Psychopharmacol 2:513-524, 1979.
5. **Weinberger DR**, Torrey EF, Neophytides A and Wyatt RJ: Lateral cerebral ventricular enlargement in chronic schizophrenia. Arch Gen Psychiatry 36:735-739, 1979.
6. **Weinberger DR**, Torrey EF, Neophytides A and Wyatt RJ: Structural abnormalities of the cerebral cortex in chronic schizophrenia. Arch Gen Psychiatry 36:935-939, 1979.
7. **Weinberger DR**, Torrey EF and Wyatt RJ: Cerebellar atrophy in chronic schizophrenia. Lancet i:718-719, 1979.
8. Luchins DJ, **Weinberger DR** and Wyatt RJ: Schizophrenia Evidence for a subgroup with reversed cerebral asymmetry. Arch Gen Psychiatry 36:1309-1311, 1979.
9. Luchins DJ, **Weinberger DR** and Wyatt RJ: Anomalous lateralization associated with a milder form of schizophrenia. Am J Psychiatry 136:1598-1599, 1979.
10. **Weinberger DR**, Kleinman JE, Luchins DJ, Bigelow LB and Wyatt RJ: Cerebellar pathology in schizophrenia: A controlled post-mortem study. Am J Psychiatry 137:359-361, 1980.
11. Jeste DV, Wagner RL, **Weinberger DR**, Rieth KG and Wyatt RJ: Evaluation of CT scans in tardive dyskinesia. Am J Psychiatry 137:247-248, 1980.
12. **Weinberger DR**, Bigelow LB, Kleinman JE, Klein ST, Rosenblatt JE and Wyatt RJ: Cerebral ventricular enlargement in chronic schizophrenia: Association with poor response to treatment. Arch Gen Psychiatry 37:11-14, 1980.
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